WEST Search History

Hide Items | Restore | Clear | Cancel

DATE: Monday, February 02, 2004

Hide?	Set Name	Query	Hit Count
	DB=PGPB,USPT,JPAB,DWPI; PLUR=YES; OP=ADJ		
☐	L7	l1 same NFkB	1
☐	L6	l4 and NFkB	1
☐	L5	l4 and NFK	0
☐	L4	TRADE alpha	3
☐	L3	L1 and (NFK beta or NFK)	3
☐	L2	L1 same (NFK beta or NFK)	0
☐	L1	TRADE or TRADE alpha	173357

END OF SEARCH HISTORY

WEST Search History

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DATE: Monday, February 02, 2004

Hide?	Set Name	Query	Hit Count
		DB=USPT,PGPB,JPAB,DWPI; PLUR=YES; OP=ADJ	
☐	L5	(TNF receptor member associated death protein) and @pd > 20021106	0
☐	L4	(TRADE alpha or TRADE beta) and @pd > 20021106	0
☐	L3	(L2 and (cell proliferation or cell death or apoptosis)) and @pd > 20021106	540
☐	L2	(TRADE) and @pd > 20021106	29319
☐	L1	(TNF Receptor member Associated with Death protein) and @pd > 20021106	0

END OF SEARCH HISTORY

factor receptor signaling pathway leading to ***NFkB*** activation.
AU Kuno, Kouji; Sukegawa, Kazuko; Ishikawa, Yuji; Orii, Tadao; Matsushima, Kouji (1)
CS (1) Dep. Pharmacol., Cancer Res. Inst., Kanazawa Univ., Takara-Machi 13-1, Kanazawa 920 Japan
SO International Immunology, (1994) Vol. 6, No. 8, pp. 1269-1272. ISSN: 0953-8178.
DT Article
LA English
AB A recent report has suggested that tumor necrosis factor (TNF) utilizes acid sphingomyelinase (SMase) pathway to activate ***NFkB*** (Schulze et al. 1992. Cell 71:765). To directly investigate the role of acid SMase in IL-1 and ***TNF*** ***receptor*** -mediated signal transduction, we examined the ability of Niemann-Pick disease (NPD) type A fibroblasts, which are deficient in acid SMase, to induce IL-8 gene expression through activating ***NFkB*** . Unexpectedly, IL-1-alpha and TNF-alpha efficiently induced IL-8 production and IL-8 mRNA in NPD type A fibroblasts as in normal fibroblasts. Furthermore, activation of ***NFkB*** was also induced in NPD type A fibroblasts in response to IL-1-alpha and TNF-alpha stimulation to a similar extent as in normal fibroblasts. These results provide evidence that acid SMase is not essential in IL-1 and ***TNF*** ***receptor*** signaling leading to ***NFkB*** activation as well as the cytokine gene activation which is regulated by ***NFkB*** .

L17 ANSWER 29 OF 30 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
AN 92156130 EMBASE
DN 1992156130
TI Mechanisms of tumor necrosis factor action.
AU Schutze S.; Machleidt T.; Kronke M.
CS Inst. fur Med. Mikrobiologie/Hygiene, Technische Universitat Munchen, Trogerstr. 4a,8000 Munchen 80, Germany
SO Seminars in Oncology, (1992) 19/2 SUPPL. 4 (16-24). ISSN: 0093-7754 CODEN: SOLGAV
CY United States
DT Journal; Conference Article
FS 026 Immunology, Serology and Transplantation
029 Clinical Biochemistry
LA English
SL English
AB Tumor necrosis factor (TNF) is able to induce a great diversity of cellular responses via modulating the expression of a number of different genes. The multitude of TNF activities may be explained by both structural and functional heterogeneity in ***TNF*** ***receptors*** as well as by a diversification of postreceptor signal transduction pathways. Purification of ***TNF*** ***receptors*** has revealed two major, distinct binding proteins (TR60 and TR80). TR60 seems to be an essential component for TNF signaling; the functional role of TR80 remains to be elucidated. The pathway of postreceptor signal transduction involves phospholipase A2, a phosphatidylcholine-specific phospholipase C, protein kinase C, and other serine/threonine and tyrosine-specific protein kinases with as yet unknown function. At the receiving end of TNF signaling, induction of gene expression is mediated through activation of nuclear transcription factors, such as ***NFkB*** , AP-1, IRF-1, and NF-GMa.

L17 ANSWER 30 OF 30 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 1991:38574 BIOSIS
DN BR40:15554
TI TNF INDUCED ***NFKB*** -HIV-1-LTR ACTIVATION CAN BE EFFICIENTLY BLOCKED
BY AN ANTI- ***TNF*** - ***RECEPTOR*** P60 ANTIBODY.
AU KRUPPA G; MEICHLE A; THOMA B; SCHEURICH P; PFIZENMAIER K; KROENKE M
CS CLINICAL RES. GROUP, MAX-PLANCK SOCIETY, GOSSLERSTR. 10D, 3400 GOETTINGEN,
FRG.
SO SEVENTH INTERNATIONAL LYMPHOKINE WORKSHOP, SAN ANTONIO, TEXAS, USA,
OCTOBER 1-5, 1990. LYMPHOKINE RES. (1990) 9 (4), 573.
CODEN: LYREDH. ISSN: 0277-6766.
DT Conference
FS BR; OLD
LA English

=>

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NEWS 5 SEP 29 DISSABS now available on STN
NEWS 6 OCT 10 PCTFULL: Two new display fields added
NEWS 7 OCT 21 BIOSIS file reloaded and enhanced
NEWS 8 OCT 28 BIOSIS file segment of TOXCENTER reloaded and enhanced
NEWS 9 NOV 24 MSDS-CCOHS file reloaded
NEWS 10 DEC 08 CABA reloaded with left truncation
NEWS 11 DEC 08 IMS file names changed
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NEWS 13 DEC 09 STN Entry Date available for display in REGISTRY and CA/CAplus
NEWS 14 DEC 17 DGENE: Two new display fields added
NEWS 15 DEC 18 BIOTECHNO no longer updated
NEWS 16 DEC 19 CROPU no longer updated; subscriber discount no longer available
NEWS 17 DEC 22 Additional INPI reactions and pre-1907 documents added to CAS databases
NEWS 18 DEC 22 IFIPAT/IFIUDB/IFICDB reloaded with new data and search fields
NEWS 19 DEC 22 ABI-INFORM now available on STN
NEWS 20 JAN 27 Source of Registration (SR) information in REGISTRY updated and searchable
NEWS 21 JAN 27 A new search aid, the Company Name Thesaurus, available in CA/CAplus

NEWS EXPRESS DECEMBER 28 CURRENT WINDOWS VERSION IS V7.00, CURRENT
MACINTOSH VERSION IS V6.0b(ENG) AND V6.0Jb(JP),
AND CURRENT DISCOVER FILE IS DATED 23 SEPTEMBER 2003
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=> FIL BIOSIS EMBASE CAPLUS
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=> s TRADE alpha
L1 3 TRADE ALPHA

```
=> s TRADE
L2      26507 TRADE

=> s l2 and NFK
L3          0 L2 AND NFK

=> s l2 and NFKB
L4          0 L2 AND NFKB

=> s l2 and NF-K
L5          0 L2 AND NF-K

=> s l2 and signaling pathway
L6          1 L2 AND SIGNALING PATHWAY

=> d bib abs

L6  ANSWER 1 OF 1  BIOSIS  COPYRIGHT 2004 BIOLOGICAL ABSTRACTS
INC. on STN
AN  2002:245633 BIOSIS
DN  PREV200200245633
TI  Mechanisms of ageing: Public or private?.
AU  Partridge, Linda [Reprint author]; Gems, David
CS  Department of Biology, University College London, Gower Street, London,
    WC1E 6BT, UK
    l.partridge@ucl.ac.uk
SO  Nature Reviews Genetics, (March, 2002) Vol. 3, No. 3, pp. 165-175. print.
    ISSN: 1471-0056.
DT  Article
    General Review; (Literature Review)
LA  English
ED  Entered STN: 17 Apr 2002
    Last Updated on STN: 17 Apr 2002


=> dup rem l1
PROCESSING COMPLETED FOR L1
L7          3 DUP REM L1 (0 DUPLICATES REMOVED)

=> d bib abs 1-
YOU HAVE REQUESTED DATA FROM 3 ANSWERS - CONTINUE? Y/(N):y

L7  ANSWER 1 OF 3  CAPLUS  COPYRIGHT 2004 ACS on STN
AN  2001:598038 CAPLUS
DN  135:175423
TI  TRADE molecules and uses related thereto
IN  Wood, Clive; Chaudhary, Divya; Long, Andrew
PA  Genetics Institute, Inc., USA
SO  PCT Int. Appl., 173 pp.
    CODEN: PIXXD2
DT  Patent
LA  English
FAN.CNT 1
    PATENT NO.      KIND  DATE         APPLICATION NO.  DATE
    --------------- ----  -------- --------------- --------
PI  WO 2001058954   A2  20010816     WO 2001-US4238  20010209
    WO 2001058954   A3  20020321
    WO 2001058954   C2  20030116
        W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,
           CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR,
           HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT,
           LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU,
           SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU,
           ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
        RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY,
           DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF,
           BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
    US 2002068696   A1  20020606     US 2001-780532  20010209
    EP 1254176      A2  20021106     EP 2001-909036  20010209
        R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
           IE, SI, LT, LV, FI, RO, MK, CY, AL, TR
    JP 2004501604   T2  20040122     JP 2001-558100  20010209
PRAI US 2000-181922P  P   20000211
    US 2000-182148P   P   20000214
    WO 2001-US4238    W   20010209
AB  The present invention relates, at least in part, to methods of modulating
    proliferation and apoptotic state of cells using agents that modulate the
    expression and/or activity of TRADE family polypeptides. In addn., the
    invention provides two novel members of the TRADE family of mols.

L7  ANSWER 2 OF 3  BIOSIS  COPYRIGHT 2004 BIOLOGICAL ABSTRACTS
INC. on STN
AN  2000:408685 BIOSIS
DN  PREV200000408685
TI  TRADE: A novel TNF-receptor family member.
AU  Long, Andrew J. [Reprint author]; Bourque, Karen [Reprint author];
    Chaudhary, Divya [Reprint author]; Haga, Hisanori; Tada, Hideaki; Burgess,
    Paul [Reprint author]; Whitters, Matthew [Reprint author]; Tan, Xiang Yan
    [Reprint author]; O'Hara, Denise [Reprint author]; Fitz, Lori [Reprint
    author]; Beier, David; McCoy, John [Reprint author]; Collins, Mary
    [Reprint author]; Shibayama, Shiro; Wood, Clive R. [Reprint author]
CS  Genetics Institute, Inc., Wyeth Ayerst Research, Cambridge, MA, 02140, USA
SO  Scandinavian Journal of Immunology, (June, 2000) Vol. 51, No. Supplement
    1, pp. 64. print.
    Meeting Info.: 8th International TNF Congress, Conference on Tumor
    Necrosis Factor and Related Molecules Scientific Advances and Medical
    Applications. Trondheim, Norway. May 14-18, 2000.
    CODEN: SJIMAX. ISSN: 0300-9475.
DT  Conference; (Meeting)
    Conference; Abstract; (Meeting Abstract)
LA  English
ED  Entered STN: 27 Sep 2000
    Last Updated on STN: 8 Jan 2002

L7  ANSWER 3 OF 3  BIOSIS  COPYRIGHT 2004 BIOLOGICAL ABSTRACTS
INC. on STN
AN  1993:206467 BIOSIS
DN  PREV199395107692
TI  Kinetic studies during enzyme hydrolysis of potato and cassava starches.
AU  Gorinstein, Shela
CS  Dep. Pharmaceutical Chem., Sch. Pharmacy, Hebrew Univ. Jerusalem, Fac.
    Med., P.O. Box 12065, Jerusalem 91120, Israel
SO  Starch, (1993) Vol. 45, No. 3, pp. 91-95.
    CODEN: STARDD. ISSN: 0038-9056.
DT  Article
LA  English
ED  Entered STN: 23 Apr 1993
    Last Updated on STN: 24 Apr 1993
AB  The hydrolysis of raw potato and cassava starches by bacterial
    alpha-amylase depends on the time of action, temperature and on the
    specific starch involved. The molecular weight of the  ***trade***
     ***alpha***  -amylase (Termamyl 60L), determined by SDS-PAGE, was found
to
    be 55-65 kDa. The properties of alpha-amylase such as kinetic parameters,
    inhibition, stability, and thermostability were studied. The constants
    K-m and maximum reaction rate V-max for alpha-amylase were fitted to
    Michaelis-Menten models with these two starches. Differences in response
    of potato and cassava starches to hydrolysis by Termamyl 60L can explain
    differences found in K-m and V-max values and inhibition properties.


=> d his

    (FILE 'HOME' ENTERED AT 17:56:59 ON 02 FEB 2004)

    FILE 'BIOSIS, EMBASE, CAPLUS' ENTERED AT 18:00:21 ON 02 FEB 2004
L1          3 S  TRADE ALPHA
L2      26507 S TRADE
L3          0 S L2 AND NFK
L4          0 S L2 AND NFKB
L5          0 S L2 AND NF-K
L6          1 S L2 AND SIGNALING PATHWAY
L7          3 DUP REM L1 (0 DUPLICATES REMOVED)

=> s TNF-receptor
L8      7917 TNF-RECEPTOR

=> s l8 and trade
L9          3 L8 AND TRADE

=> dup rem l9
PROCESSING COMPLETED FOR L9
L10         3 DUP REM L9 (0 DUPLICATES REMOVED)

=> s l10 not l1
L11         1 L10 NOT L1

=> d bib abs

L11 ANSWER 1 OF 1  BIOSIS  COPYRIGHT 2004 BIOLOGICAL ABSTRACTS
INC. on STN
AN  2000:377034 BIOSIS
DN  PREV200000377034
TI  ***TRADE***  , a novel  ***TNF***  ***receptor***  superfamily
    member, induces apoptosis and activates NFkappaB and Jnk.
AU  Chaudhary, Divya [Reprint author]; Long, Andrew J. [Reprint author];
    Bourque, Karen [Reprint author]; Adams, David H.; Hubscher, Stefan G.;
    Towler, Paul [Reprint author]; Potts, Douglas [Reprint author]; Wood,
    Clive R. [Reprint author]
CS  Genetics Institute, Inc., Wyeth Ayerst Research, 87 Cambridge Park Drive,
    Cambridge, MA, 02140, USA
SO  Scandinavian Journal of Immunology, (June, 2000) Vol. 51, No. Supplement
    1, pp. 33. print.
    Meeting Info.: 8th International TNF Congress, Conference on Tumor
    Necrosis Factor and Related Molecules Scientific Advances and Medical
    Applications. Trondheim, Norway. May 14-18, 2000.
    CODEN: SJIMAX. ISSN: 0300-9475.
DT  Conference; (Meeting)
    Conference; Abstract; (Meeting Abstract)
LA  English
ED  Entered STN: 6 Sep 2000
    Last Updated on STN: 8 Jan 2002


=> s l8 and NFkappaB
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L12 197 L8 AND NFKAPPAB

=> dup rem l12
PROCESSING COMPLETED FOR L12
L13 188 DUP REM L12 (9 DUPLICATES REMOVED)

=> d bib abs

L13 ANSWER 1 OF 188 CAPLUS COPYRIGHT 2004 ACS on STN
AN 2003:879044 CAPLUS
DN 139:394859
TI Tumor Necrosis Factor Receptor-associated Factor 2 (TRAF2)-deficient B Lymphocytes Reveal Novel Roles for TRAF2 in CD40 Signaling
AU Hostager, Bruce S.; Haxhinasto, Sokol A.; Rowland, Sarah L.; Bishop, Gail A.
CS Interdisciplinary Program in Immunology, Department of Pediatrics, University of Iowa, Iowa City, IA, 52242, USA
SO Journal of Biological Chemistry (2003), 278(46), 45382-45390
CODEN: JBCHA3; ISSN: 0021-9258
PB American Society for Biochemistry and Molecular Biology
DT Journal
LA English
AB CD40 function is initiated by tumor necrosis factor (***TNF***) ***receptor*** -assocd. factor (TRAF) adapter proteins, which play important roles in signaling by numerous receptors. Characterizing roles of individual TRAFs has been hampered by limitations of available exptl. models and the poor viability of most TRAF-deficient mice. Here, B cell lines made deficient in TRAF2 using a novel homologous recombination system reveal new roles for TRAF2. We demonstrate that TRAF2 participates in synergy between CD40 and B cell antigen receptor signals, and in CD40-mediated, TNF-dependent IgM prodn. We also find that TRAF2 participates in the degrdn. of TRAF3 assocd. with CD40 signaling, a role that may limit inhibitory actions of TRAF3. Finally, we show that TRAF2 and TRAF6 have overlapping functions in CD40-mediated NF-.kappa.B activation and CD80 up-regulation. These findings demonstrate previously unappreciated roles for TRAF2 in signaling by ***TNF*** ***receptor*** family members, using an approach that facilitates the anal. of genes crit. to the viability of whole organisms.
RE.CNT 56 THERE ARE 56 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> s l8 and NFkappaB and train
L14 0 L8 AND NFKAPPAB AND TRAIN

=> s l12 and train
L15 0 L12 AND TRAIN

=> s TRAIN-R
L16 3 TRAIN-R

=> dup rem l16
PROCESSING COMPLETED FOR L16
L17 2 DUP REM L16 (1 DUPLICATE REMOVED)

=> s l17 and l12
L18 0 L17 AND L12

=> d bib abs l17

L17 ANSWER 1 OF 2 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED.
on STN
AN 1999106137 EMBASE
TI Primary nocturnal enuresis: Current.
AU Cendron M.
CS Dr. M. Cendron, Dartmouth-Hitchcock Medical Center, Lebanon, NH, United States
SO American Family Physician, (1 Mar 1999) 59/5 (1205-1214).
Refs: 42
ISSN: 0002-838X CODEN: AFPYAE
CY United States
DT Journal; Article
FS 007 Pediatrics and Pediatric Surgery
027 Biophysics, Bioengineering and Medical Instrumentation
028 Urology and Nephrology
037 Drug Literature Index
LA English
SL English
AB Primary nocturnal enuresis sometimes presents significant psychosocial problems for children and their parents. Causative factors may include maturational delay, genetic influence, difficulties in waking and decreased nighttime secretion of antidiuretic hormone. Anatomic abnormalities are usually not found, and psychologic causes are unlikely. Evaluation of enuresis usually requires no more than a complete history, a focused physical examination, and urine specific gravity and dipstick tests. Non-pharmacologic treatments include motivational therapy, behavioral conditioning and bladder- training exercises. Pharmacologic therapy includes imipramine, anticholinergic medication and desmopressin. These drugs have been used with varying degrees of success.

=> d bib abs l12 2

L12 ANSWER 2 OF 197 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 2003:279222 BIOSIS
DN PREV200300279222
TI Endothelial activation by angiotensin II through ***NFkappaB*** and p38 pathways: Involvement of ***NFkappaB*** -inducible kinase (NIK), free oxygen radicals, and selective inhibition by aspirin.
AU Costanzo, Antonio; Moretti, Francesca; Burgio, Vito Lelio; Bravi, Cristina; Guido, Francesco; Levrero, Massimo [Reprint Author]; Puri, Pier Lorenzo
CS Laboratory of Gene Expression, Fondazione A. Cesalpino, Universita' degli Studi di Roma "La Sapienza", Viale del Policlinico 155, 00161, Roma, Italy levrero@ifo.it; levmax@tin.it
SO Journal of Cellular Physiology, (June 2003) Vol. 195, No. 3, pp. 402-410. print.
CODEN: JCLLAX. ISSN: 0021-9541.
DT Article
LA English
ED Entered STN: 11 Jun 2003
Last Updated on STN: 11 Jun 2003
AB Angiotensin-II (AII), the dominant effector of the renin-angiotensin system, is involved in the pathogenesis of cardiovascular diseases, such as atherosclerosis. Upregulation of the adhesion molecules VCAM-1, ICAM-1, and E-selectin in endothelial cells by inflammatory cytokines through nuclear factor kappa B (***NFkappaB***) activation is implicated in formation and progression of atherosclerotic plaque. Here we show that AII induces ***NFkappaB*** -dependent transcription in primary endothelial cell lines, leading to the upregulation of ICAM-1 and VCAM-1 expression. ***NFkappaB*** activation by AII is mediated by the ***NFkappaB*** -inducing kinase (NIK), a common mediator of ***NFkappaB*** activation by inflammatory cytokines, such as TNF-alpha. However, ***NFkappaB*** stimulation by AII differs from that of TNF-alpha since a ***TNF*** - ***receptor*** associated factor 2 (TRAF-2) dominant negative mutant does not prevent AII-mediated ***NFkappaB*** activation. In analogy with TNF-alpha-dependent activation of ***NFkappaB*** , treatment with either the anti-oxidant N-acetyl cysteine (NAC) or the cyclooxygenase (COX) inhibitor acetyl salicylic acid (aspirin), but not indometacin, prevents the induction of ***NFkappaB*** -dependent transcription by AII. Thus, production of reactive oxygen species, aspirin (asp)-sensitive enzymes of the arachidonate metabolism, and NIK are common transducers of AII- and TNF-dependent pathways to ***NFkappaB*** . AII also activates the inflammatory p38 kinase in endothelial cells, an effect inhibited by exposure to either NAC or asp. Pharmacological interference of the p38 pathway, with the inhibitor SB 202190, prevented AII-mediated activation of the ***NFkappaB*** target V-CAM, without affecting degradation of IkappaBalpha. These results support a pro-inflammatory effect of the vasoactive peptide AII in endothelial cells, through at least two pathways- ***NFkappaB*** and p38-both of which are sensitive to asp and antioxidants.

=> d his

(FILE 'HOME' ENTERED AT 17:56:59 ON 02 FEB 2004)

FILE 'BIOSIS, EMBASE, CAPLUS' ENTERED AT 18:00:21 ON 02 FEB 2004
L1 3 S TRADE ALPHA
L2 26507 S TRADE
L3 0 S L2 AND NFK
L4 0 S L2 AND NFKB
L5 0 S L2 AND NF-K
L6 1 S L2 AND SIGNALING PATHWAY
L7 3 DUP REM L1 (0 DUPLICATES REMOVED)
L8 7917 S TNF-RECEPTOR
L9 3 S L8 AND TRADE
L10 3 DUP REM L9 (0 DUPLICATES REMOVED)
L11 1 S L10 NOT L1
L12 197 S L8 AND NFKAPPAB
L13 188 DUP REM L12 (9 DUPLICATES REMOVED)
L14 0 S L8 AND NFKAPPAB AND TRAIN
L15 0 S L12 AND TRAIN
L16 3 S TRAIN-R
L17 2 DUP REM L16 (1 DUPLICATE REMOVED)
L18 0 S L17 AND L12

=> s TNF receptor
L19 7917 TNF RECEPTOR

=> s l19 and r248
L20 1 L19 AND R248

=> d bib abs

L20 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2004 ACS on STN
AN 2001:397044 CAPLUS
DN 134:362290
TI Cloning of human ***TNF*** ***receptor*** ***R248*** cDNA and its use in the treatment of immune disorders
IN Kitson, Jeremy David Alistair
PA Glaxo Group Limited, UK

SO PCT Int. Appl., 34 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2001038526	A1	20010531	WO 2000-GB4438	20001121

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

PRAI GB 1999-27681 A 19991123
AB A novel ***TNF*** ***receptor*** ***R248*** is provided which is a screening target for the identification and development of novel pharmaceutical agents which modulate the activity of the receptor and in particular modulate activation of NF.kappa.B by the receptor. The present invention provides the cDNA sequences coding for human ***TNF*** - ***receptor*** ***R248*** polypeptide. A method for identification of a substance that modulates ***TNF*** ***receptor*** activity comprises contacting a polypeptide of the invention with a test substance in the presence of a reporter whose activity is mediated by NFkB and monitoring NFkB mediated activity.
RE.CNT 9 THERE ARE 9 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> s TRAIN receptor
L21 1 TRAIN RECEPTOR

=> d bib abs

L21 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2004 ACS on STN
AN 1999:194264 CAPLUS
DN 130:232854
TI TRAIN: A cysteine-rich member of the tumor necrosis factor receptor family
IN Tschopp, Jurg; Hession, Catherine
PA Biogen, Inc., USA
SO PCT Int. Appl., 32 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 2

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 9913078	A1	19990318	WO 1998-US19030	19980911

W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, HR, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

CA 2301173	AA	19990318	CA 1998-2301173	19980911
AU 9892303	A1	19990329	AU 1998-92303	19980911
AU 738688	B2	20010927		
EP 1012282	A1	20000628	EP 1998-944859	19980911

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI

JP 2002505843	T2	20020226	JP 2000-510863	19980911
US 2003219860	A1	20031127	US 2002-303502	20021122

PRAI US 1997-58631P P 19970912
US 1998-84422P P 19980506
WO 1998-US19030 W 19980911
US 2000-522436 B1 20000309
AB A new member of the tumor necrosis factor receptor family, called TRAIN, is identified and a cDNA encoding it is cloned and characterized. The protein may be a target for use in treatment of tumors (no data).
RE.CNT 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> s l1 and NFkappaB
L22 0 L1 AND NFKAPPAB

=>

---Logging off of STN---

=>
Executing the logoff script...

=> LOG Y

COST IN U.S. DOLLARS	ENTRY	SINCE FILE SESSION	TOTAL
FULL ESTIMATED COST		83.72	84.98

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS) SINCE FILE TOTAL

	ENTRY	SESSION	
CA SUBSCRIBER PRICE		-2.77	-2.77

STN INTERNATIONAL LOGOFF AT 18:14:37 ON 02 FEB 2004

---Logging off of STN---

END

Unable to generate the STN prompt.
Exiting the script...